

Pre-term Spontaneous Unscarred Uterine Rupture; A Case Report and Review

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| ARTICLE INFO | ABSTRACT |
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| <p>Article type: Case Report</p> <hr/> <p>Article history: Received: 28-May-2014 Accepted: 22-June-2014</p> <hr/> <p>Keywords: Preterm uterine rupture Pregnant woman Spontaneous uterine rupture</p> | <p>Introduction: Spontaneous uterine rupture, a life-threatening condition for both parturient and their fetus, often tends to occur during labour, particularly in those with a scarred uterus (rare in unscarred ones). The diagnosis must be born in mind in case of a pregnant woman presenting with shock, abdominal pain and diminished fetal heart rate. Any additional preoperative evaluation seems redundant as it only wastes golden surgical time.</p> <p>Case: The case we intend to present is a multigravid woman (G3L2) in 28th week of gestation with an unscarred uterus. She had cardiac surgery six months earlier Atrial Septal Defect (ASD) device closure with Percutaneous Coronary Intervention (PCI). She presented with severe dyspnea, diminished lung sounds in the right lower zone, chest pain at rest, and radiating pain to her right shoulder from 12 hours earlier. No FHR was detected by auscultation with Doppler. She had generalized abdominal tenderness and rebound tenderness.</p> |

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Introduction

Patient

A woman with two prior vaginal deliveries presented at the 28th week of gestation to the maternity clinic of "Imam Reza Training hospital" with the chief complaint of severe dyspnea and chest pain at rest and radiating pain to her right shoulder from 12 hours before. She was pale and alert in appearance, unable to lie down due to respiratory distress had a fundal height of 28 weeks, and no Fetal Heart Rate (FHR) was detected by auscultation with Doppler.

There was not a history of uterus surgery, trauma or other risk factors for uterine rupture, namely diagnostic or therapeutic intrauterine intervention, any report of vaginal bleeding or labour pains in patients past medical history. Noteworthy is an angiographic Atrial Septal Defect (ASD) closure six months earlier, after which she received plavix for one month and aspirin for the next four months.

On physical exam, lung sounds were diminishing on auscultation whereas there was no detection of any heart murmur and she had generalized abdominal tenderness and rebound tenderness. Vital signs included: BP (Blood Pressure)=135/85 mmHg, PR (Pulse Rate)=138/min, RR (Respiration Rate)=23/min and spo₂= 91%. She had pelvic guarding on touch, with both flanks dull on percussion. The decreased right

lung sounds on auscultation of the lungs were found. In her paraclinical investigations, fetal heart rate could not be detected on ultrasonography. Lab data revealed a platelet count of 120000 and a Hematocrit of (25%).

With a provisional diagnosis of suspected abruption, and on our cardiologist's recommendation, it was decided for her to undergo cesarean section for the termination of her pregnancy. After the placement of standard monitoring and arterial line, initial volume resuscitation was commenced with Lactated Ringers' solution. Also, five units of Red Blood Cell (RBC) packs-Iso group and Iso Rhesus factor (RH) were reserved at the same time. Her acute hemorrhage was estimated around two or three, in accordance with American College of Surgeons' classification. The patient then underwent a rapid sequence of induction with two mg/kg ketamine, 50 microgram fentanyl and one mg/kg succinylcholine. As she lost consciousness, Sellick's maneuver, was applied followed by a traumatic intubation. General Anesthesia (GA) was maintained with Isoflurane (0/75%, 1/0%) oxygen and volume expansion with both Ringers and blood when available.

During laparotomy, 2500 cc of blood mixed with amniotic fluid and a clot of 800 cc volume were

drained, which led to the normalization of lung sounds on both sides.

Surgical findings included a full thickness rupture of the uterine fundus in coronal plane between both cornua (at least with ten cm in length) and an intraperitoneal dead fetus. There was no sign of placental insertion in the abdominal-pelvic cavity, nor was there any indication of any uterine anomaly and placental abruption, or active bleeding in ruptured site.

The fundus was covered with a discoid clot with a 25 cm diameter. A hysterectomy was not performed the fundus myometrium was repaired in two layers (first with running lock sutures and the second layer with mattress sutures separately), and was performed the uterine serosa, was sutured continuously; after that, double tubal ligation using the parkland method.

Following the procedure, the abdominal wall was closed, and the patient was transferred to the Gynecology ward after regaining consciousness. The post-operative course was uncomplicated, with the transfusion of two packed cells. The patient was discharged on her third post-operative day.

Discussion

Uterine rupture can vary in incidence among nations, ranging from one to 93 of 9908 (0.010%-1.07%). This can be justified in accordance with socioeconomic status, education level, and other similar influential parameters.

Uterine rupture can seriously jeopardize both fetal and maternal life (1, 2). There is a wide range of predisposing factors, including, previous cesarean delivery or myomectomy (3, 4), congenital uterine anomaly, uterine tumors, past direct or indirect uterine trauma (motor vehicle accident), violence (gunshot wound), placentation (5, 6, 7, 8), grand multiparity, uterine over-distention (9, 10), induced labour (oxytocin or PG) (11, 12), cornual or extra-uterine pregnancy (13), dystocia, intrauterine manipulation (breech) (14), high fundal pressure (15, 16) and unknown (17).

However rupture of unscarred uterus is a rare event involving 1:16,000 deliveries (18). The probable causes in reported cases are external injuries, induction of labor, high birth order, cephalo-pelvic disproportion, placenta accreta, fundal pressure, abruption, cocaine abuse, and history of intrauterine intervention causing perforation (19-26).

On the other hand, atrial septal defects are usually well tolerated during pregnancy. In a study by Actis, miscarriage, preterm delivery, and cardiac deterioration occurred more frequently in patients who did not undergo surgical correction of their defect prior to pregnancy (25). Generally, decisions concerning pregnancy in this group should be made on an individual basis considering functional status, pulmonary hypertension, and the presence of additional cardiac lesions (26). Fetal bradycardia is the most common and characteristic clinical manifestation of

uterine rupture. Variable or late decelerations may precede bradycardia, but there is no fetal heart rate pattern pathognomonic of rupture. Furthermore, fetal heart rate changes alone have low sensitivity and specificity for diagnosing uterine rupture (27).

Maternal manifestations are variable. Uterine rupture should always strongly be considered if constant abdominal pain and signs of intraabdominal hemorrhage are present. Vaginal bleeding is not a cardinal symptom, as it may be modest, despite major intra abdominal hemorrhage. However, case reports and series indicate that pain may not be present in sufficient intensity, character, or location to suggest uterine rupture, (1, 4) and pain may be partially or completely masked by regional analgesia. Furthermore, although hemorrhage is common, the signs and symptoms of intraabdominal bleeding in cases of uterine rupture, especially those cases not associated with prior surgery, may be subtle (28). Other potential clinical manifestations include maternal tachycardia, hypotension ranging from subtle to severe (hypovolemic shock), cessation of uterine contractions, loss of station of the fetal presenting part, uterine tenderness, and change in uterine shape. Intuitively, loss of integrity of the uterine wall should be associated with a reduction in intrauterine pressure, but case series where an intrauterine pressure catheter was in place at the time of rupture have generally not observed pressure changes significantly different from laboring patients without rupture (29, 30). An increase in baseline intrauterine pressure was seen in four of 39 patients in one series (29). But in our case with severe dyspnea at rest, decreased right lower lung zone sounds, and past history of ASD repair without vaginal bleeding, more preoperative evaluations and lab tests or consultations could be dangerous because time to referring the patient to the operating room and emergency surgery for saving the patient's life might be lost. In our case we had a desired pregnancy with no manipulation and there was no placenta accreta, couvelaire uterus, any signs of placental abruption or history of connective tissue disorder like Ehler Danlos syndrome (31). It thus makes us speculate that this rupture could have been due to some inherent weakening in the uterine myometrium, which became more profound in third pregnancy due to repeated stretching from previous pregnancies.

Conclusion

This case re-emphasizes that rupture uterus can present in many different ways and high suspicion is required for timely intervention to prevent maternal morbidity and mortality so time-consuming pre operative diagnosis should be reduced.

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